

Epidemics scenarios in the “Romantic network”

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Abstract

Background: The structure of sexual contacts, its contacts network and its temporal interactions, play an important role in the spread of sexually transmitted infections. Unfortunately, that kind of data is very hard to obtain. One of the few exceptions is the “Romantic network” which is a complete structure of a real sexual network of a high school. In terms of topology, unlike other sexual networks classified as scale-free network, it does not correspond to any model network, as the authors of the work concluded based in many network measures. Regarding the temporal structure, several studies indicate that relationship timing can have effects on diffusion through networks, as relationship order determines transmission routes.

Methodology: With the aim to check if the particular structure, static and dynamic, of the Romantic network is determinant for the propagation of an STI in it, we perform simulations in two scenarios: the static network where all contacts are available and the dynamic case where contacts evolve in time. In the static case, we compare the epidemic results in the Romantic network with some paradigmatic topologies. We further study the behavior of the epidemic on the Romantic network in response to three variants. First, we investigate the effect of any individual, belonging to the network, having a contact with an external infected subject. Second, we explore the influence of the degree of the initial infected. Third, we analyze the effect of the variability of contacts per unit time. Finally, we consider the dynamics of formation of pairs in the Romantic network and we study the propagation of the diseases in this dynamic scenario.

Conclusions: Our results suggest that the scale-free network displays indeed a substantial difference with the Romantic network. On the other side, while this real network can not be labeled as a Watts-Strogatz network, it is, regarding the propagation of an STI, very close to one with high disorder. Additionally, we find in relation to the three epidemic variations: (i) the effect that any individual contacting an external infected subject is to make the network closer to a full connected one, (ii) a higher contact degree of the initial infected accelerates outbreaks (earlier peak time and higher epidemic probability) and (iii) the epidemic impact is proportional to the numbers of contacts per unit time. Finally, our simulations confirm that relationship timing affects, but strongly lowering, the final outbreak size. Besides, shows a clear correlation between the average degree and the outbreak size over time.

Introduction

The progress in medical and biological sciences by the end of sec XIX eventually led to the identification of the organisms responsible for the diseases, making possible the systematization of the mathematical description of epidemics. While first attempt to mathematical model epidemics is situated probably at 1760 with Daniel Bernoulli and his study of vaccination on the spread of smallpox [1], was Hamer who introduced the idea of mass action [2] to model epidemics. Then Kermack and McKendrick used that idea to evolve into the present form of the susceptible-infective-removed (SIR) model [3], and its variants, so ubiquitous in the nowadays theoretical description of epidemics [4, 5]. As a mass action or mean field model, the SIR model (and its SIS, SIRS, etc, variants) assumes that all classes are superimposed in

space¹. However, in the last decade, it became clear that the transmission of a disease can be influenced by the contact network [6, 7]. Such network depends on the structure of interactions among the individuals, which in turn may depend on the peculiar disease. Several articles have addressed the disease propagation problem on networks using paradigmatic examples like small-world [8] or scale-free networks [9]. The high heterogeneity of some extreme topologies lead to important consequences as the persistence of epidemics for any value of the spreading rate in an infinite scale-free network² [11, 12]. General results regarding the threshold, size and distribution of epidemic bursts were given for small-world networks in one and two dimensions [13].

While there have been recent efforts to incorporate human behavior into disease models (see ref [14] for a review), to implement disease-spreading agent based models, including attributes at real human populations sizes, it is yet a very difficult task [15]. Before that level of sophistication became eventually a not so hard enterprise, the study of the spreading of hypothetical diseases in real networks can be of great help. Until recently, the complete description of a real network—at least one which could be related with a specific disease—was something that seemed not feasible. Consequently, the only information at hand was statistical data like the degree distribution of the network associated with the disease. That is the case of the Sweden study [16], in which a questionnaire addressing sexual behavior was completed by 4781 subjects. Unfortunately, that kind of questionnaire not only is subjected to bias³ [17], but does not allow us to build the real network behind the individuals, because the subjects represent a small sample of a larger population, the links of which are not known.

However, since some years ago, the picture of a small, but real, sexual and romantic network is available. The “Romantic Network”, as called by the authors [18], is one of a few examples of a complete network⁴, so simulation of epidemics on it can be very important, even considering its restricted validity.

Moreover, information on the dynamic formation of links is available for the Romantic network [19], which makes this particular example more interesting. If we take the static network and run a simulation of an epidemic, with some values of infectivity and infection time, at the end we can have let say 10% of the subjects infected. Now, if we take into account the dynamics of formation of links, what will be the final result? In other words, how different will be the final outcome from the static scenario? There is no answer to that question without doing a simulation in the dynamic network and that is one of the principal aims of the present contribution.

Thus, in the present contribution we implement and analyze computer simulations of the evolution of an hypothetical sexually transmitted infection (STI) in the Romantic network. From such simulations it is possible to evaluate the risk and impact of a potential epidemic in this and similar networks. The analysis is based on the specific structure of that network and it is compared to several well known model networks, which are potentially representative of human interactions.

Once the network is established and known, one is faced with different schematic ways of conducting the disease spreading simulations. Consider an infected node with some other susceptible nodes connected to it. The different ways in which the contagion can go from this node to its neighbors can be schematically divided in three cases: at each time step (which can represent a day for example) it can be through only one link, through some of them simultaneously, or through all of them in the same period. Among the three possibilities and for matters of simplicity we chose the first one (one contact per time step) for all the simulations of the Results section, leaving one specific sub-section to discuss the other possible

¹Even in the more elaborated case of space distributed classes (Fisher’s equations) it is locally assumed the superposition of them.

²The reason comes from the non epidemic threshold of an infinite-variance degree distribution of such infinite network, however human networks dubiously present such a extreme behavior; for more details see Ref [10].

³For our purposes, the most relevant questions are about the number o partners in the last year and in lifetime, the last one is particularly strong biased.

⁴There are other two networks more recently available and made from real data: the Likoma network [20] and the Internet sex network of Brazil [21]. However, from the figures of the first one it is not possible to code the network as we did with the romantic network. The second one was obtained indirectly from posts to a forum and restricted to sex commerce contacts. For a broad picture of the available human networks and how the data were obtained see Ref [22].

choices.

Different from the usual approach of mass action models and static networks, several recent articles [23–27] have addressed the problem of disease propagation from the perspective of dynamic formation of links. As was emphasized in those contribution we believe that the timing present in all social networks is a key factor regarding the flow of information (disease, rumors, etc) that goes through them. Therefore one section of our contribution is devoted to this subject.

Materials and Methods

Empirical network

In 2004 the first picture of an almost complete sexual network of a social group was published [18]. This network, called by the authors “Romantic and Sexual Network” emerged from data taken from the National Longitudinal Study of Adolescent Health, a longitudinal study of students in grades 7-12 in US. Among the 140 schools included in the study, 14 had saturated field-settings⁵. From those, one of the two largest ones ($N \approx 1000$) was used to depict the aforementioned network. The data was obtained after home interviews covering a period of 18 month, where students were asked to identify their sexual or romantic partners from a list of the student attending their school. “Jefferson High School” is the fake name of the only public high school in “Jefferson City”, a non-identified mid-sized mid-western town of US, with a homogeneous (all-white, mostly working class students) and isolated community. Participated in the in-school survey 90% of the school roster and a total of 873 completed the questionnaires. After data was collected a network of 573 nodes emerged, from which probably the most remarkable feature is a ring-like component involving 288 students. The 288 nodes component emerged as a result of superimposing all contacts among the students during the 18 month period, see Fig. 1B.

However, taking into account the temporal sequence, James Moody published a dynamic version of this network in his site [19]. In order to see the evolution of an hypothetical infection both in the static and dynamic network, the animated figure cited above was broken into its 45 frames, each one corresponding to a period of 12 days, which for simplicity we assume evenly distributed in time. The nodes and links between them were identified and converted to input data for the simulation of the epidemic models. Each frame taken individually looks very different from the superposition of all of them (the giant static component). To give an idea of how it looks like, we show in Fig. 2 two frames at times $t_1 = 10$ and $t_2 = 34$, which represent times of low and high activity in the network, respectively. Therefore, it could not have been guessed before the picture was finished, nor by the researchers, neither by the participants. Besides, we manually mapped the graphical representation of the giant component (static and dynamic versions) and checked with Pajek [28] that our transcription gave the same graphical representation than the original one.

Artificial networks

For the artificial networks we use the Watts-Strogatz (W-S) [29] and Krapivsky-Redner (K-R) [30] models, which are good candidates for representing human relations. They cover many scenarios from ordered to disordered, and from homogeneous to heterogeneous, respectively, by means of one control parameter. Besides, they allow an easy control of the average degree.

The W-S networks are implemented starting from a ring of 288 nodes linked up to first neighbors. We let the disorder parameter p to have several values running from 0 (the original ordered ring) to 1 (a complete random network), avoiding the rupture of the component, see Fig. 1A. So all degrees of disorder are probed besides the normally accepted small-world values ($p \approx 0.01$). For the heterogeneous K-R

⁵In “saturated” cases **all** students of the school took the in-home interviews, which ask to nominate their partners, so complete sexual and/or romantic networks could therefore be constructed with these data.

networks we use the redirection procedure (see Fig. 1C): at every time step, a new node is added to the system and an earlier node i is selected uniformly, as a possible target for attachment. With probability $1 - r$ a directed edge from the new node to i is created; with probability r the edge is redirected to the ancestor node j of node i . Starting from two connected nodes we let the network evolve until it reaches a 288 nodes component. This model leads eventually to a power-law degree distribution —when it grows to large sizes— with degree exponent $\gamma = 1 + \frac{1}{r}$, which can be tuned to any value equal or larger than 2.

The real network is bipartite (145 males and 143 females), but for all the epidemic dynamics in the real and artificial networks we consider all nodes of the same kind, because we assume them as non directional networks, without distinction between links. Apart from the size, the most relevant feature for the implementation of model networks that have to mimic the largest connected component of the Romantic network, is the average degree $\langle k \rangle$. Its value is $\langle k \rangle = 2$, so all artificial networks were build having this same value.

Simulation of epidemics

The epidemics in the networks are studied by computational simulations of the SIR model. For each specific network topology and realization —in which each vertex represents a person, with links representing the sexual relation—, one individual is randomly chosen to be the first infected, leaving the rest in the susceptible state. That is the initial state i.e., one node in the I state and the other 287 nodes in the S state. The simulation proceeds as follow: at each time step (which could represent one day or one week), each infected person chose a partner randomly from the list of subjects connected with him/her. If the chosen node is in infected or removed state, nothing happens, but if is in susceptible state it becomes infected with probability β ⁶. Such procedure mimics a sexual contact of two persons in which one of them has an STI. The simulation step is completed after all nodes have contacted one of its partners, so we assume that the sexual activity is homogeneous among the population and independent of the number of partners. We will discuss other options later. Once infected, the subject remains so for a fixed period τ —being potentially contagious to its list of contacts as the dynamics proceeds. After that period, the infected individual is removed (or becomes permanent immune). The process is repeated until no infected individual remains in the population. For a given situation, the disease parameters β and τ are fixed and have the same value for all subjects.

The time step, as we mentioned before, can represent one day or one week or another reasonable time period. If it is one day, that means that every subject is having one intimate or sexual contact per day, which is probably an overestimation. However, the simulation step could be assigned to a week which can underestimate the danger of the epidemic. Anyway, that frequency related to sexual activity is absorbed in the $\beta\tau$ number, and a broad range of them will be tested in this presentation.

For the static scenario, the Romantic and artificial networks are characterized by the following quantities relevant for the SIR model: the final size of the epidemic, $R(\infty)$, the maximum value of infected subjects or epidemic peak, I_{max} , the time to arrive to that peak, t_p , and the epidemic probability. All of them were obtained computing those realizations that ended up with more than 5% of infected subjects. It is indeed the ratio between the number of those realizations and the total what we use to define the probability to observe an epidemic. The number of different realization for each kind of network (and for each value of $\beta\tau$) is two thousand, which corresponds to ten different graphs for each kind of network and two hundred different random seeds of the random number generator (i.e. different initial infected subject) used to simulate the infection.

⁶There are three random processes in the simulations which are implemented by the use of a pseudo-random number generator in the computer (we use Marsaglia random generator [31]): the selection of the first infected among the N (288) nodes, the selection of one of the partners of each infected node at each time step, and the stochastic process of infection with probability β . The two first ones are like throwing a dice, while the last one is implemented in a Monte Carlo (MC) procedure: by generating a random number uniformly in the $[0,1)$ interval, and comparing with β , if the number is smaller (or equal) than β , the infection occurs, otherwise not. Besides, a MC procedure is used previously to generate the W-S and K-R networks.

Other possibilities in relation to the Romantic network are explored: external field, degree of the initial infected, and rules of interaction.

By external field, B , we mean the probability that a subject within the static empirical network interacts with an outsider, who is assumed to be infected. Therefore, the chosen one, selected at random from the entire population, is infected with probability β . Obviously, $B = 0$ is equivalent to the normal dynamics without external excursions, while $B = 1$ means that at each time step someone certainly encounters an infected outsider. $B\beta$ is then the likelihood of an spontaneous infection in the network.

Then, we probe the influence of the initial infected. In particular, we want to see such influence regarding the degree of the node, i.e. the number of connections of the initial infected subject. The rationale for this is that someone with many links should have more potential of infection than someone with only one link. We think this is a very important question to elucidate before going to epidemics in real situations. In order to test that, we define the initial infected individual according with its degree, from the minimum value $k_i = 1$ to the maximum $k_i = 9$, comparing between them and with the random election of the initial infected. When k_i has its maximum value there is only one option for the initial infected, because only one subject in the static empirical network has such degree. Then, as the degree of the initial infected goes down, i.e. exploring lower values of k_i , we have more options for the initial infected (there are more individuals with lower values of connectivity). In those cases we average over several initial choices of subjects. Besides, in all cases, we average over many initial random seeds, so even in the one possibility initial case ($k_i = 9$) it represents the average of different possible “histories”, starting from that situation.

In real social dynamics the frequency of interactions is not uniformly distributed, i.e. one person could visit one or many of its contacts during a fixed period of time. In order to capture this feature in the epidemic models we face different possibilities when an infected subject interacts with its contacts; we call them rules of interaction. One: from all its contact, only one is chosen randomly at each time step. Fraction: a subset of its links is selected at random to contact this set at the same time step. All: all connected vertices are contacted at the same time step.

The dynamic network brings a new scenario. The inclusion of the dynamic formation of links modulates one of the basic properties of the network, the degree. In order to characterize the evolution of the dynamic network we measure (using the snapshots of the animated gif) the degree of each node over time and we use this data to obtain the dynamic degree distribution, $P_t(k)$. This distribution represents the probability that a randomly chosen node, at a given time t , has degree k . From this quantity, we follow the changes of the average degree over time, $\langle k \rangle_t$. Since at different times, many sub-graph are disconnected from the giant component, another relevant measure is the size of the largest connected component, G . Lastly, we characterized the spread of the epidemic in this scenario. For this, we measure the fraction of infected nodes I .

Results

Static Scenario

Romantic network vs artificial networks

Before presenting the results let us make two important comments. In the graphical representation of the Romantic network originally presented in Bearman et al. paper [18], apart from the giant component of 288 nodes, there are many small components ranging from pairs, triplets up to ten nodes which gives a total of 573 students. However any infection started at any one of those small components will be restricted to it. Consider the whole network of 573 elements results in a theoretical renormalization of the giant component results by the 288/573 ratio, which we have checked (see Fig. 3A). The second comment refers to Figure 3B, which clearly shows that different pairs of β and τ values, but with the same value of $\beta\tau$, collapse to the same point. This is well known from the differential equation of the

classical SIR model [5], however we want to explicitly show that it holds too for numerical simulations on static networks. So the statistical properties of disease outbreaks depend only on the product $\beta\tau$ which, along with the degree distribution $P(k)$, defines the theoretical value of the basic reproductive number R_0 . Therefore, in what follows, our results will be shown in term of $\beta\tau$ which, as expected, appears to be a good parameter to characterize all of them.

From a general perspective, we observe that, except for the K-R networks with $r \approx 1$ (the most heterogeneous networks), many topologies give similar epidemic results provided that $\beta\tau$ is below 4. This can be concluded from the comparison between the Romantic and the W-S networks, as can be seen in Fig.4, and the K-R for $r < 1$, as shown in Fig.5. Moreover, if we consider a small group of topologies, the W-S with large p (≈ 1) and the K-R with small r (≈ 0), the behavior of the results, specially $R(\infty)$ and the epidemic probability (Figs. 4,5 C,D) are very similar to the corresponding ones from the Romantic network, up to values of $\beta\tau$ equal to 8. Considering individual realizations, the displayed error bars in Fig.4A-C and Fig.5A-C correspond to the Romantic network and give an idea of the observed dispersion associated to the small size of it.

Besides, we observe that the mean field (all mixed) scenario is the worst possible one from the epidemic point of view. Any other situation, i.e., any other network structure in which not everybody is potentially connected with anybody, even the K-R heterogeneous networks yield a less unfavorable picture. The only exception is the K-R network with $r = 1$ (which would give a scale-free network for an infinite system) but for values of $\beta\tau$ less than 1.

Comparing the outcome between the different networks and the Romantic one, it is possible to see that the last one does not fit into any of the model we have tried. Nevertheless, within the uncertainties of the fluctuation associated with the small (288 nodes) networks considered here, an epidemic in a random network obtained with large p (≈ 1) is statistically undistinguished from an epidemic in the Romantic network. On the other side, the topology that diverges more from the Romantic network (and from all the others) is the K-R network with $r = 1$. This particular value of r gives a power law distribution network with exponent $\gamma = 2$ (scale-free) in the limit of large sizes. In the present case, however, due to the small size of the networks, results in a star topology, very different from the ring-like type of the Romantic network.

External field

The Romantic network was constructed taking into account all the sexual contacts of the surveyed school but excluding any possible contact outside the school. This section is devoted to explore the effect that a possible contact of a member of the network with an infected subject outside of it can have in the epidemic dynamics inside the school. This effect is controlled by the parameter B which represent the probability of the external excursion to happen.

As can be see in Fig. 6, the effect of the external field makes the outcome of the infection, measured by the total number of infected individuals as a function of $\beta\tau$, closer to what is expected from the standard SIR model. This suggests that the practical effect of the external field is to make the network closer to a full connected one.

Degree of the initial infected

The results, presented in Fig. 7, are a little subtle to interpret. Looking at Fig. 7B,C (I_{max} and $R(\infty)$ respectively) we are tempted to conclude that the influence of whom is the initial infected one, if it is more or less connected, is irrelevant —which we have to admit is not obvious. However, the other two figures (Fig. 7A,D), representing the time to the infection peak and specially the last one, i.e., the epidemic probability, show a clear divergence from low ($k_i = 1$) to high ($k_i = 9$) degree of the initial infected. At $\beta\tau = 4$ for example, we clearly see in Fig. 7C that, independently of who was the initial infected, the average removed subjects at the end is around 10%. Nevertheless, Fig. 7D remind us that in the

low degree case that situation happens only in 30% of the instances, contrasting with the 90% observed in the high degree case. Therefore, as expected, the degree of the initial infected is a key factor in the aftermath of a disease propagation in the Romantic network.

Rules of interaction

The three different measures shown in Fig. 8 all point out to the same general trend: from rule “one” to rule “all” we gradually approach the mean field all mixed case, which remains as the upper limit. Even the extreme “all” rule is below that limit, because subjects in the romantic network are not fully connected.

Dynamic Scenario

Measurements

The Fig. 9A displays the degree distribution at different times. The mere inspection of that figure makes clear that any measure we can think of for networks will give very different values if we applied to an individual frame, instead of the static network that results from the superposition of all the frames. That can be appreciated in Fig. 9B, where we compare the degree distribution at three different times around the most active frame, with the static network degree distribution. As time goes by, we have: for $t < 15$ the interactions are only pair-like, with no connection between different pairs at the same time; the time window $30 < t < 40$ is where the activity increases to its maximum at $t = 35$; for $t > 40$ the degree distribution returns to low values. This is an extremely important point, because unlike some proposed models for the spread of epidemics in dynamic networks [25, 26, 32], this scenario can not be represented by an asymptotic degree distribution.

Greater is the effect over the average degree, $\langle k \rangle_t$, which has a peak value $\langle k \rangle = 1$ at $t \approx 35$, but most of the time it is much lower than the static value $\langle k \rangle = 2$ (Fig. 9C). Probably the most important effect of such measure—in fact the reason of that difference—is that in all frames, many individuals have no contacts at all ($k = 0$). Following the behavior of random networks [33], we observe that the variation of the average degree directly influences the giant component size, G .

Another important measurement, related with the dynamic formation of links, is the fraction of time that each individual participates in the sexual activity, which is represented in Fig. 9D. That figure shows, for example, that while the majority of the students ($\sim 90\%$) participates less than 45% of the time, a small fraction of the them ($\sim 10\%$) is active during more than 55% of the total time.

Dynamic disease

The first result (see Fig. 10A,F) is remarkable: for the wide interval of $\beta\tau$ values explored, the final size of the epidemic is always much smaller than the corresponding static results⁷. Evidently, no epidemic should be observed in this scenario. In fact, we can see that only for infection times τ close to the whole duration of the dynamics, there are some effects over the susceptible population, but very weak indeed. Therefore, it is interesting to consider an extreme epidemic case, the SI epidemic dynamics, which means a lifetime infection time⁸, so the relevant time is the duration of the dynamics. This is what is presented in Figures 10 with parameters $\tau = 540$ and $\beta = 0.2$, except in Fig. 10A where all possible values of these two parameters are explored (up to the extreme value $\beta = 1$, which means a certain infection every time an S-I contact is tried). Even with this extreme setup the total number of infected individuals at the end is under 15%. The average number of infected however, considered all the possible students as the

⁷For the comparison to be possible we use the same unit of time (a day) for the time step in both cases; each of the 45 frames in the dynamic scenario lasts 12 days, which gives a total of 540 days. So, in the static scenario we use a total of 540 time steps.

⁸Some diseases, notoriously infection by HPV virus, have this kind of dynamics where τ is equal to the lifetime.

initial infected one is not bigger than 5% (see Fig. 10B and 10C). Just to take the situation to the very extreme, Fig. 10D shows the number of final infected as a function of the number of initially infected, and we see that with more than 50 over 288 initial infected (17%) the final proportion of infected is below 80%. All together these results strength the key role of the dynamic formation of pairs which bring a different scenario for modeling epidemics in networks.

Besides, in the dynamic network, the total number of infected individual is strongly affected by the initial condition, as can be seen in Fig. 10E. In this case, who is the initial infected is very important, not because of its personal activity, but due to the particular sequence in which the individuals connect to each other. It is the whole sequence which matters, not the particular initial subject. From the dynamics we see that the number of infected subjects grows substantially from $t > 360$.

Figure 10F shows the comparison between the static and dynamic Romantic network within the SI disease. The difference is remarkable. The static network, in any of the transmission rules, is much more efficient for the disease propagation than the dynamic version.

Discussion

In this contribution we addressed the problem of STI epidemics in a real network (the so called “Romantic or Jefferson School network”) in two aspects. First, to compare epidemics in this network with epidemics in model networks to see whether the later ones (or which one of them) may represent a real network. Particularly, if the disease propagation is sensitive enough to details in the topology. The second aspect, the effect of the dynamic formation of links and how this can affect the epidemic dynamics, is far more relevant, specially because of the conclusion we have drawn.

Regarding the static network and the comparison between the Romantic network and the paradigmatic ones, the maximum incidence and the final size or prevalence of the epidemic are sensitive to the topology for relative large values of $\beta\tau$ (> 5). For smaller values of $\beta\tau$ (< 4), the different topologies —except the high heterogeneous ones— are hard to distinguish in terms of epidemic outcome. Even more if we consider the wide dispersion of the overall results due to the small size of the networks (bars in Fig.4,5).

Homogeneous networks, like the Watts-Strogatz with $p \approx 1$ or Krapvisky-Redner with $r \approx 0$ are statistically similar between them and with the Romantic network in terms of epidemic outcome. This is valid when $\beta\tau$ is not very large and it is due to the peculiar average value of the degree ($\langle k \rangle = 2$) which makes all the structures ring or tree like, as can be seen by direct eye inspection of Fig. 1 (A-rightmost, B, and C-leftmost). The main difference between these artificial realizations and the real one is its ring. However is an only and relative large ring. Among all artificial static networks tested in this contribution, we can conclude that the Watt-Strogatz network with $p \approx 1$ (random network with $\langle k \rangle = 2$) is undistinguished from the Romantic network in terms of disease spreading, for values of $\beta\tau$ up to 8.

A marginal conclusion is that any network structure, in which not everybody is potentially connected with anybody, including the K-R heterogeneous networks, give a less drastic scenario than the mean field all mixed result. The only exception is the K-R network with $r \approx 1$ (and for $\beta\tau < 1.5$) because it approaches a scale-free network.

Considering the external field, degree of the initial infected, and rules of interactions, our results suggest that the epidemic dynamics is affected by all these variants.

Regarding the external field, the effect that individuals contacting infected external subjects has is to make the network closer to a full connected one.

Concerning the degree of initial infected (or the sexual activity of the starter of the infection) its importance is confirmed in the Romantic network. At $\beta\tau = 4$ for example, the chances of a burst involving more than 5% of the subjects triples when going from the less to the most connected as the initial infected one. The higher the degree of the initial infected the shorter the time to reach the peak of the infection.

Our results on contacts frequency (rules of interactions) confirm that the spread of an epidemic not only depends on the available number of contacts, but also on the frequency of contacts with them [34], and as the frequency increases the results approach the mean field results from below.

Going to the dynamic network situation, that is considering the sequence of connections and disconnection between the subjects, we arrive at very interesting conclusions. We have shown with simulations (the only possible tool in this case) that the dynamic network will always cause less severe epidemics. Because not only there are periods in which we only see small isolated components (at those times the infection can not jump from one component to the others), but the time windows in which they are connected (and not all at the same time) are relatively short. In other words, relationship timing directly influences the course of the epidemics [24].

Clearly, the propagation of a disease that depends on the links, which are created but for definite time intervals, will be strongly affected by such dynamical process. We have seen that the average degree in the dynamic case has a peak of value $\langle k \rangle = 1$, but usually is much lower than the static value $\langle k \rangle = 2$. Besides, in the dynamic case the distribution of interaction times, absent in the static case, is crucial for disease propagation.

By testing an hypothetical extreme epidemic situation, the Susceptible-Infective model with maximum infection probability, we obtained a final number of infected around 15% in the worst case (starting the infection with the most connected subject). However, the average number of infected subjects, over all the possible choices of the initial infected student is not bigger than 5%. Putting together, these results make apparent the key role of the dynamic formation of pairs, which brings a different scenario from previous results in static networks. Remarkably, in the dynamic network the total number of infected individuals is strongly affected by the initial condition. In this case, who is the initial infected is important, not due to its personal activity, but because of the particular sequence in which the individuals connect between them. It is the whole sequence which matters, not the particular subject.

Moreover, comparing figures Fig. 9C and Fig. 10E we see a clear correlation between them. Thus, the dynamic of the largest component is related with the evolution of the fraction of infected subjects. Like a random network, the giant component follows a close relation with the average degree, as can be seen comparing $\langle k \rangle_t$ and G/N in Fig. 9C. This leads us to conclude that the fraction of infected people is related to the dynamics of the average degree, which can be important for public health.

Additionally, we observe that the static network, in any of the transmission rules, is much more efficient for the disease propagation than the dynamic version. The effect of the dynamic formation (and break) of links is quite clear: A disease that depends on the structure of the links which are not available all the time, but in very specific time windows, will be influenced by them; but in a “desirable way”, because the worst scenario is always the static one, when all links are available all the time.

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Figure Legends

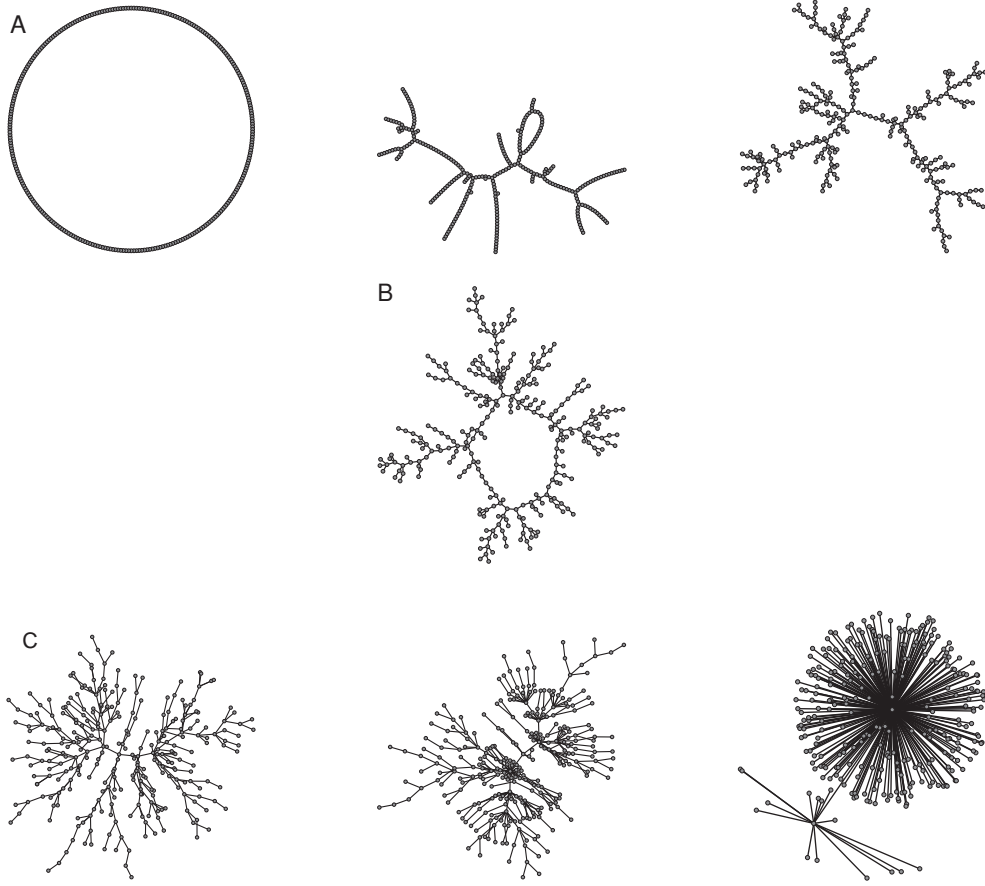


Figure 1. Networks realizations used in this work: (A) Watts-Strogatz: from left to right, $p = 0$ (regular), $p = 0.1$, and $p = 1$ (random); (B) Romantic; (C) Krapivsky-Redner: from left to right, $r = 0.1$, $r = 0.5$, and $r = 1$. All of them have the same number of vertices, $N = 288$, and the same average degree, $\langle k \rangle = 2$. Figures generated with PAJEK [28].

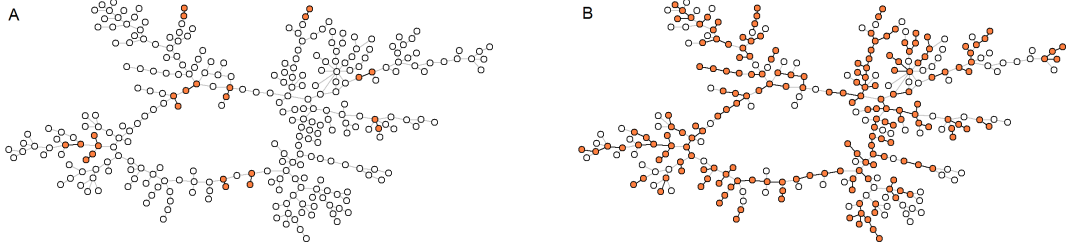


Figure 2. Snapshots of the network taken from frames at times (A) $t_1 = 10$ and (B) $t_2 = 34$. The hatched vertices correspond to the interacting individuals. Figures generated with PAJEK [28].

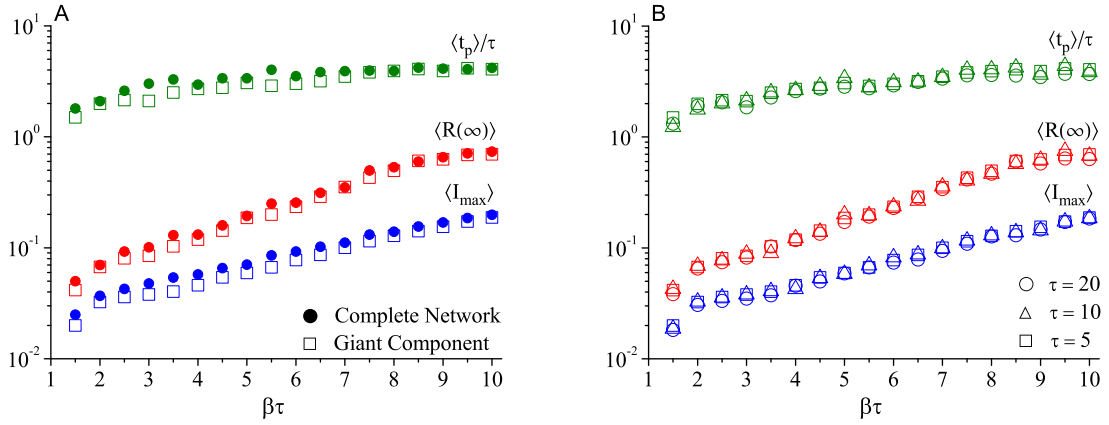


Figure 3. Behavior of the disease spread in the (static) Romantic network regarding: (A) Comparison between giant component and complete network (scaled by $573/288$, i.e. the *complete network/giant component* size relation) and (B) the $\beta\tau$ product. Each point of the curves is the result of an average over 200 independent runs (different seed of the random number generator).

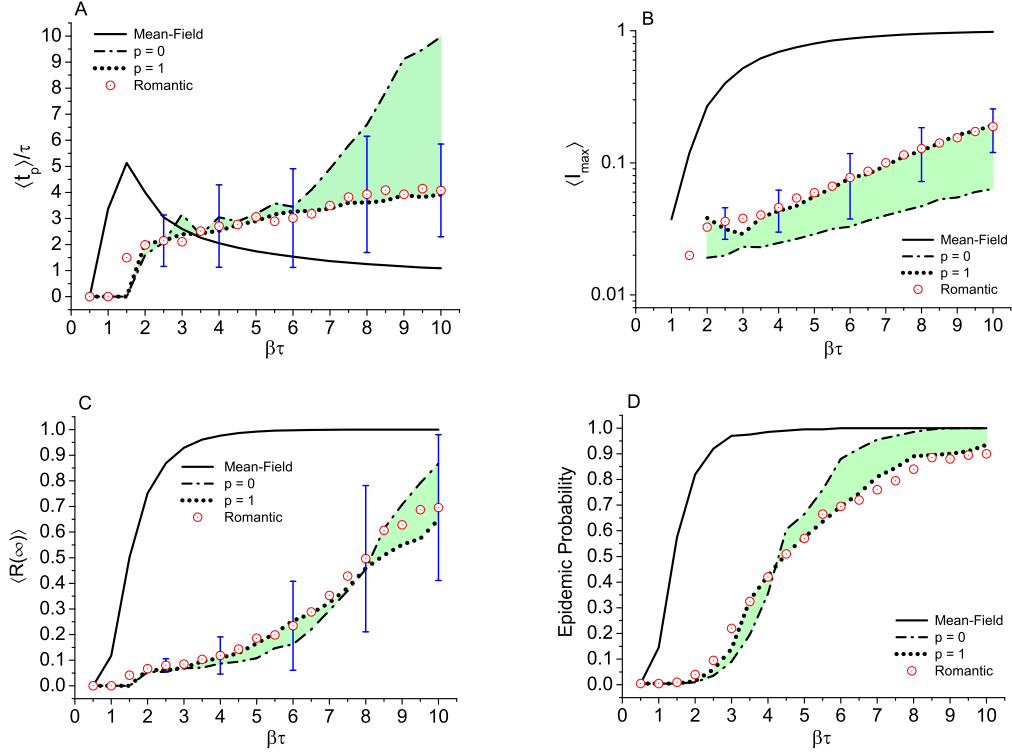


Figure 4. Epidemic simulation of the SIR model in different topologies: Watts-Strogatz, Romantic, and fully connected networks. Results for (A) time to the epidemic peak, (B) maximum number of infectives, (C) total number of removed or final prevalence, and (D) epidemic probability, defined as the fraction of realizations that end up with more than 5% of the population affected. Each point of the curves is the result of an average over 200 independent runs on 10 different graphs (except for the Romantic network which is only one graph). Error bars, displayed only at some points, correspond to the standard deviation over the 200 runs in the Romantic network case. Green shadows help to visualize the variation of the outcome due to the progressive disorder in the W-S networks.

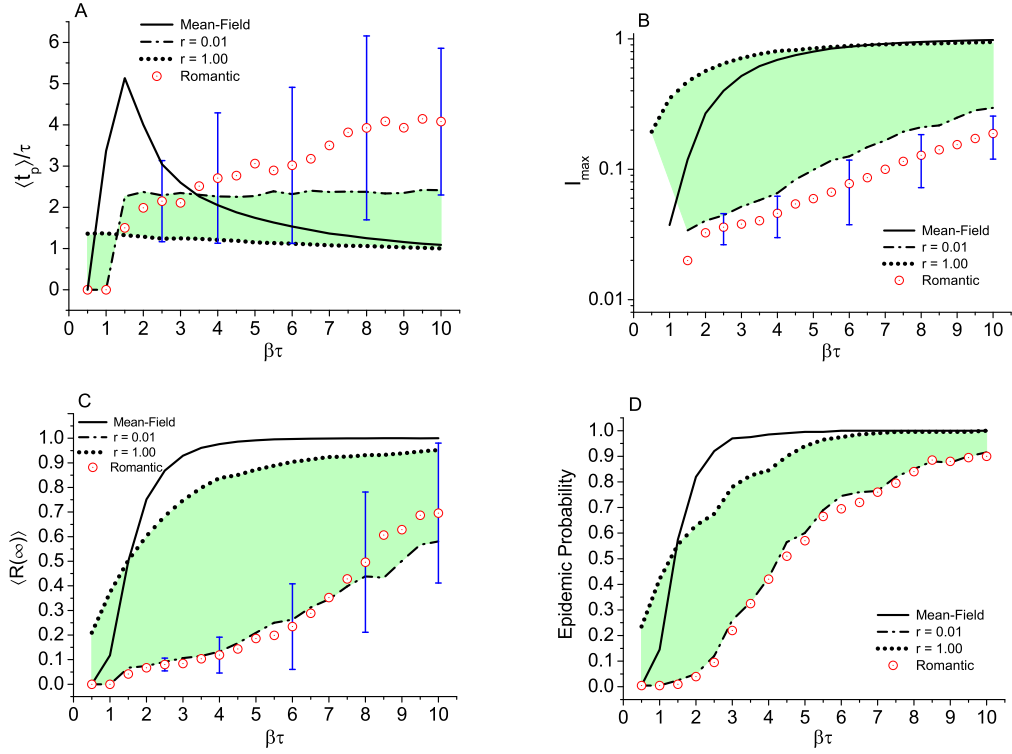


Figure 5. Epidemic in different topologies: Krapivsky-Redner, Romantic, and fully connected networks. Results for (A) time to the epidemic peak, (B) maximum number of infectives, (C) final prevalence, and (D) epidemic probability. Number of runs, average per point, and error bars as in Fig. 4. Green shadows help to visualize the variation of the outcome due to the progressive heterogeneity in the K-R networks.

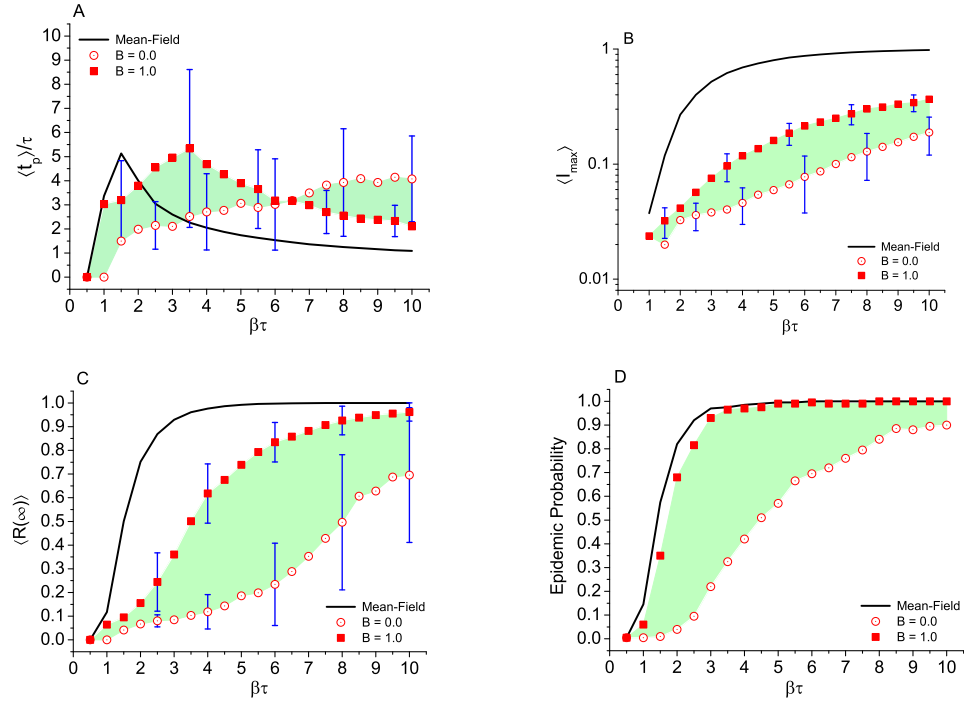


Figure 6. Effect of the “external field”, B , applied to the Romantic network: Results for (A) time to the epidemic peak, (B) maximum number of infectives, (C) final prevalence, and (D) epidemic probability. Comparison with standard mean field results in a network of the same size. Number of runs, average per point, and error bars as in Fig. 4. Green shadows help to visualize the variation of the outcome as increasing the B parameter.

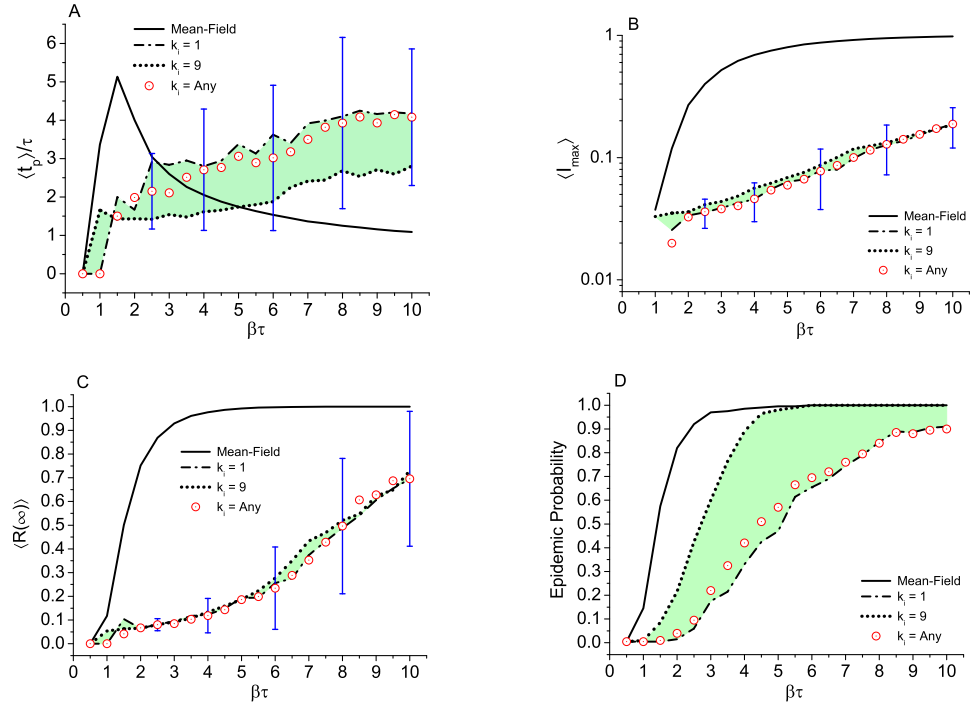


Figure 7. Effect of degree of the subject chosen to be the initial infected in the Romantic network: Results for (A) time to the epidemic peak, (B) maximum number of infectives, (C) final prevalence, and (D) epidemic probability. Comparison with standard mean field results in a network of the same size. Number of runs, average per point, and error bars as in Fig. 4. Green shadows help to visualize the variation of the outcome as response to the degree variation of the initial infected subject.

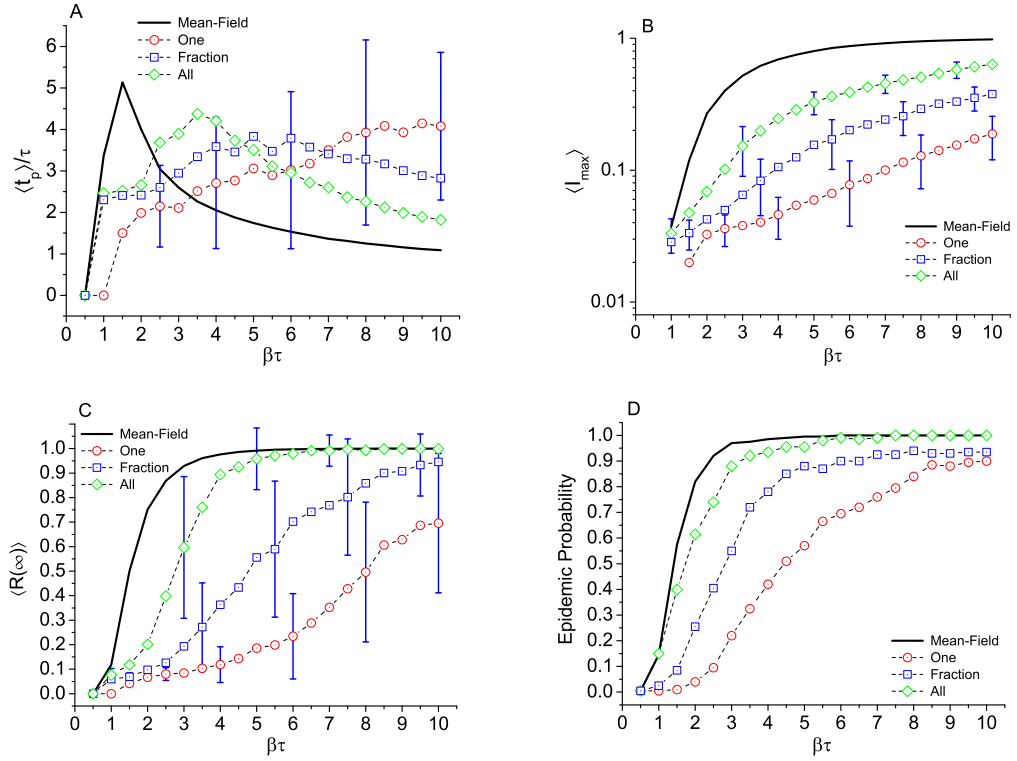


Figure 8. Effect of the different rules of interaction on: Results for (A) time to the epidemic peak, (B) maximum number of infectives, (C) final prevalence, and (D) epidemic probability. Comparison with standard mean field results in the same network. Number of runs, average per point, and error bars as in Fig. 4.

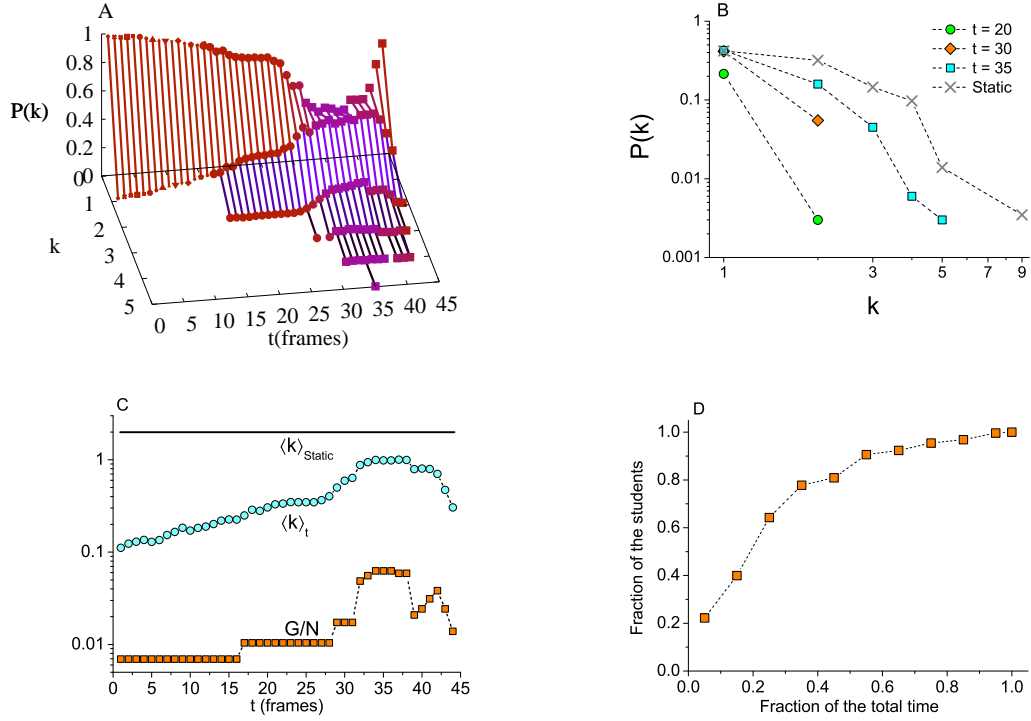


Figure 9. Characterization of the dynamic network. (A) Evolution of the degree distribution of the giant component of the Romantic network, when the frames are taken separately. (B) Degree distribution at three selected times ($t = 20, 30, 35$) compared with the static degree distribution. (C) Average degree $\langle k \rangle_t$ and largest connected component size G/N (normalized by $N = 288$) as a function of time. (D) Cumulative distribution of interaction times, as a fraction of the total time.

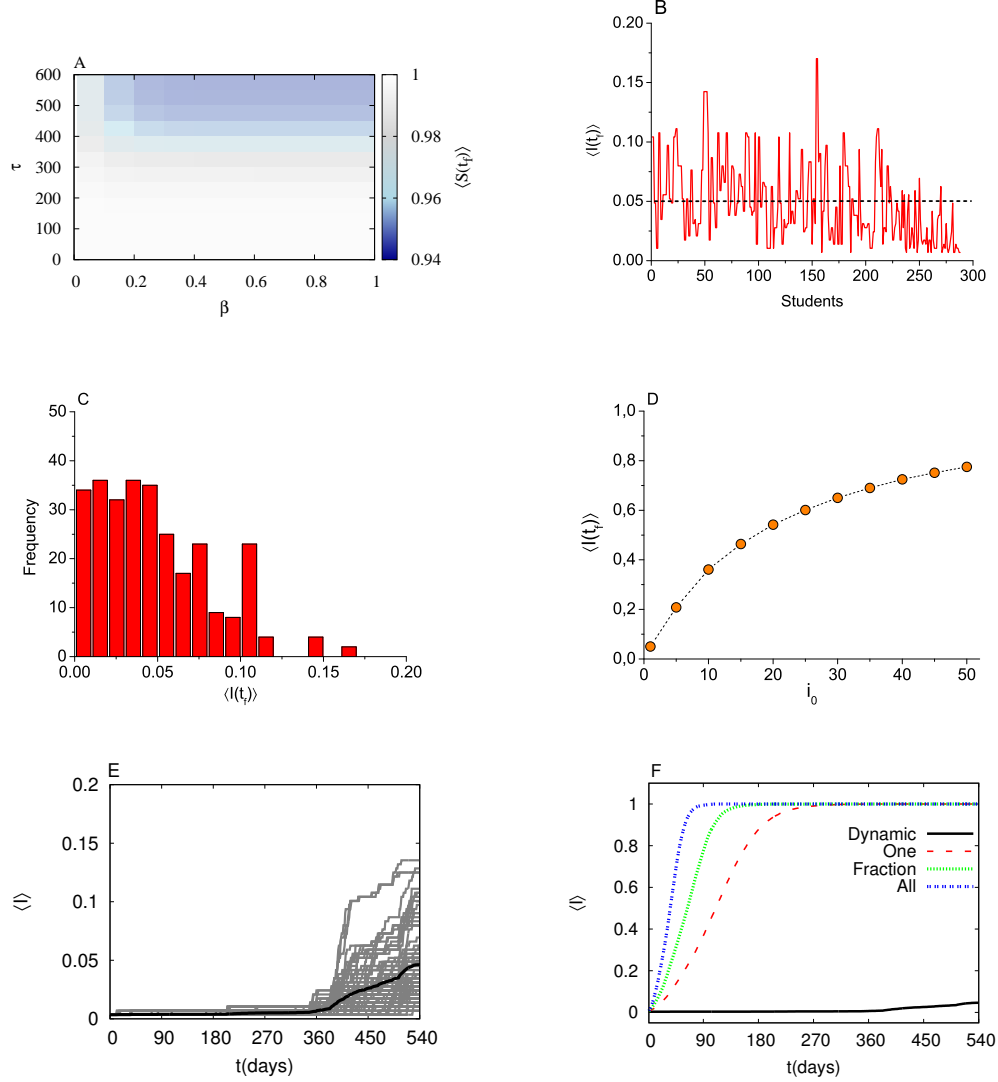


Figure 10. Epidemics simulations on the “Dynamic Romantic network”: (A) Final fraction of non-infected subjects $\langle S(t_f) \rangle$ as a function of the infective time τ and the infection probability β (white region means almost no infection); (B) distribution of infection size (fraction of total ever infected) depending on the initial infected subject; (C) histogram of the distribution (B); (D) average infection size (fraction of total ever infected) vs the number of initial infected subjects; (E) fraction of infected subjects vs time; different runs with different seeds in gray, average in black; (F) average number of infected subjects vs time, comparison between dynamic network and static network with different dynamics rules. Each point of the curve is the result of an average over 200 independent runs. In figures B-F $\tau = 540$ and $\beta = 0.2$ (notice however that figure A shows that for such value of τ the dependence on β is weak above 0.2).